

CHAPTER 17

Blood Pressure

KEY TEACHING POINTS

- There are two methods of blood pressure measurement: the traditional auscultatory method (using the stethoscope to detect Korotkoff sounds) and the oscillometric method (automated machines). Only the auscultatory method detects pulsus paradoxus, pulsus alternans, and pulsus bisferiens. The oscillometric method, in contrast, reduces observer biases and avoids the error of the auscultatory gap.
- Hypotension is an ominous finding in hospitalized patients, predicting increased mortality and adverse outcomes.
- A difference of more than 20 mm Hg in the systolic blood pressure of the arms is abnormal, suggesting either subclavian steal syndrome (if the patient has symptoms of vertebrobasilar ischemia) or aortic dissection (if the patient has acute chest pain).
- In patients with known cardiomyopathy, a *narrow* pulse pressure (i.e., proportional pulse pressure less than 0.25) increases probability of low cardiac output. In patients with the murmur of aortic regurgitation, a *wide* pulse pressure (80 mm Hg or more) increases the probability of moderate-to-severe regurgitation.
- When measuring postural vital signs (i.e., comparing supine and standing positions), hypovolemia is likely if there is *either* a pulse increment of 30/minute or more *or* the patient cannot stand because of dizziness.

I. INTRODUCTION

Systolic blood pressure is the maximal pressure within the artery during ventricular systole, diastolic blood pressure is the lowest pressure in the vessel just before the next systole, and pulse pressure is the difference between the systolic and diastolic values. Pulse pressure may be normal, abnormally small (narrow), or abnormally large (wide; see the section on [Abnormal Pulse Pressure](#)). The mean arterial pressure can be estimated by $(S + 2D)/3$, where S is systolic blood pressure and D is diastolic blood pressure.¹

The first person to measure blood pressure was Stephen Hales, an English clergyman of creative genius, who in 1708 directly connected the left crural artery of a horse to a 9-foot-tall glass manometer using brass tubes and the trachea of a goose.^{2,3} Vierordt of Germany introduced the indirect method of measuring blood pressure in 1855, based on the principle that blood pressure is equal to the amount of external pressure necessary to obliterate the distal pulse. Indirect measurements required cumbersome mechanical devices and were not widely accepted until 1896, when the Italian Riva-Rocci invented the blood pressure cuff.^{2,3}

Blood pressure was the last of the four traditional vital signs to be routinely monitored in hospitalized patients. In 1901, after Harvey Cushing first brought the blood pressure cuff to America and encouraged its use in neurosurgical patients, most clinicians resisted using it because they believed palpation of pulse revealed much more information, including its “fullness,” “tension,” “rate,” “rhythm,” “size,” “force,” and “duration”.^{4,5} Two events were responsible for clinicians eventually accepting the blood pressure cuff: (1) Korotkoff described his sounds in 1905, which allowed clinicians to easily measure systolic and diastolic blood pressure using a stethoscope, and (2) Janeway published his book *Clinical Study of Blood Pressure* in 1907, which proved that monitoring blood pressure was clinically useful. Janeway showed, for example, that the first sign of intestinal perforation or hemorrhage in patients with typhoid fever was progressive hypotension.⁶ By the time of the First World War, blood pressure was routinely recorded by most clinicians, along with the patient’s pulse rate, respiratory rate, and temperature.^{5,7,8}

II. TECHNIQUE

A. AUSCULTATORY VERSUS OSCILLOMETRIC METHODS

There are two methods of measuring blood pressure: The **auscultatory method** (the traditional method) uses a stethoscope to detect Korotkoff sounds in the brachial artery as a blood pressure cuff is slowly manually deflated. Aneroid manometers have largely replaced the original standard for this method, the mercury sphygmomanometer, because of mercury’s environmental risks and bans on its use.⁹ The **oscillometric method** analyzes pressure oscillations within the cuff itself and uses proprietary computer programs to calculate the blood pressure and display the result digitally.⁹

The auscultatory method has the advantage of being able to detect abnormalities of pulse contour, such as *pulsus paradoxus*, *pulsus alternans*, and *pulsus bisferiens*, all abnormalities missed using the oscillometric method (see [Chapter 15](#)). The oscillometric method, in contrast, has the advantages of convenience, reduced observer bias, and elimination of the auscultatory gap. (See the sections later on [Terminal Digit Preference and Auscultatory Gap](#).)

B. RECOMMENDED TECHNIQUE^{9,10}

Published recommendations for measuring blood pressure are based on the consensus opinion of expert committees who have reviewed all available scientific evidence. These recommendations, however, are designed to avoid misdiagnosis of *hypertension* and may not be as relevant to clinicians using the blood pressure cuff to diagnose other abnormalities, such as *hypotension* or abnormalities of pulse contour. (See [Chapter 15](#) and the section on Clinical Significance later in this chapter.)

The important elements of the correct technique are as follows: (1) The patient should sit in a chair with his or her back supported and should rest for at least 5 minutes before the blood pressure is measured. (2) The patient’s arm should be at the level of the heart. (3) The length of the blood pressure cuff’s bladder should encircle at least 80% of the arm’s circumference. (4) The clinician should inflate the cuff to a pressure 20 to 30 mm Hg above systolic pressure, as first identified by palpation of the distal pulse (i.e., the pulse disappears when cuff pressure exceeds systolic pressure). (5) The pressure in the cuff should be released at a rate of 2 mm Hg per second. (6) The clinician should obtain at least two readings separated by at least

30 seconds and average them; if these differ by more than 5 mm Hg, additional readings are necessary. (7) The readings should be rounded off to the nearest 2 mm Hg.

In some clinical scenarios, described in the section Findings and Their Clinical Significance, additional measurements are necessary, including those of the legs or opposite arm or measurements taken with the patient in different positions.

C. KOROTKOFF SOUNDS (AUSCULTATORY METHOD)

I. DEFINITION OF SYSTOLIC AND DIASTOLIC BLOOD PRESSURE

As the cuff is slowly deflated from a point above systolic pressure, the first appearance of sound (Korotkoff phase 1) indicates systolic blood pressure.* Clinicians have debated for decades whether the muffling of sound (Korotkoff phase 4) or disappearance of sound (Korotkoff phase 5) better indicates diastolic blood pressure, although now all experts favor using phase 5 for the following reasons: (1) in most studies, phase 5 sounds correlate better with intra-arterial measurements of diastolic blood pressure;^{14,15} (2) many persons lack phase 4 sounds;^{14,16} (3) interobserver agreement is better for phase 5 sounds than phase 4 sounds;^{14,16} and most importantly, (4) long-term observational and treatment studies correlating hypertension and cardiovascular risk events have used phase 5 sounds for definition of diastolic blood pressure.

2. PATHOGENESIS

Korotkoff sounds are produced underneath the *distal* half of the blood pressure cuff.¹⁷ The sounds appear when cuff pressures are between systolic and diastolic blood pressure, because the underlying artery is collapsing completely and then reopening with each heartbeat. The artery collapses because cuff pressure exceeds diastolic pressure; it opens again with each beat because cuff pressure is less than systolic pressure. The sound represents the sudden deceleration of the rapidly opening arterial walls, which causes a snapping or tapping sound, just like the sail of a boat snaps when it suddenly tenses after tacking in the wind or a handkerchief snaps when its ends are suddenly drawn taut.¹⁷⁻²¹ Once cuff pressure falls below the diastolic blood pressure, the sound disappears because the vessel wall no longer collapses but instead gently ebbs and expands with each beat, being held open by diastolic pressure.

The genesis of the Korotkoff sounds, therefore, is similar to the genesis of other snapping or tapping sounds produced by the sudden deceleration of other biologic membranes, such as the normal first and second heart sounds or the femoral pistol shot sounds of aortic regurgitation (see [Chapters 40 and 45](#)).

D. MEASUREMENT USING PALPATION

Even before the discovery of Korotkoff sounds, clinicians used the blood pressure cuff to measure both systolic and diastolic blood pressure.⁶ Systolic blood pressure was simply the amount of cuff pressure necessary to obliterate the pulse. Clinicians still use this technique to measure the blood pressure of hypotensive patients (a

*There are five Korotkoff phases, numbered in order as they appear during deflation of the cuff. The initial tapping sound at systolic blood pressure is phase 1; a swishing murmur is phase 2; the reappearance of a softer tapping sound is phase 3; the disappearance of the tapping and appearance of a much softer murmur ("muffling") is phase 4; and the disappearance of all sound is phase 5.² Korotkoff described only four of these sounds (phases 1, 2, 3, and 5). Ettinger added the muffling point (phase 4) in 1907.^{7,11,12} All five phases are audible with electronic stethoscopes in 40% of adults.¹³

setting when Korotkoff sounds are often too faint to hear) or to determine whether the patient has an auscultatory gap. (See the section later on the [Auscultatory Gap](#).)

To identify diastolic pressure, clinicians can use one of two methods. In the first method, the clinician applies light pressure to palpate the brachial artery just below the blood pressure cuff. As the cuff is deflated, the first appearance of a pulse indicates systolic blood pressure. As the cuff pressure decreases and approaches diastolic pressure, the pulsatile forces distending the artery distal to the cuff progressively grow, eventually causing a sudden shock to strike the clinician's fingers as the artery abruptly opens and then completely collapses with each beat. (This abrupt tapping sensation is similar to the **water hammer pulse** of aortic regurgitation.)¹⁸ At the moment the cuff pressure falls below diastolic blood pressure, the shocking sensations disappear, being replaced by a much gentler pulse, because the underlying artery no longer collapses completely between beats. The cuff pressure at this *lower limit of maximal pulsation* indicates the diastolic blood pressure.⁶

A second method requires a rigid and tightly applied cuff, so that the arterial pulsations under the cuff are actually transmitted to the manometer. As the cuff pressure decreases, the indicator needle of an aneroid manometer starts to bob with increasing amplitude, until the bobbing suddenly disappears at the moment cuff pressure falls below diastolic pressure.⁶ Many patients with tightly applied cuffs also experience a similar pounding sensation in their arm near the diastolic pressure, which abruptly disappears the moment cuff pressure falls below diastolic blood pressure.

Measurements of systolic and diastolic blood pressure by palpation differ from readings by auscultation by only 6 to 8 mm Hg or less.^{22,23}

E. POSTURAL VITAL SIGNS²⁴

When obtaining postural vital signs (i.e., comparison of measurements when the patient is supine with those when the patient is upright), clinicians should wait 2 minutes before measuring the supine vital signs and 1 minute after standing before measuring the upright vital signs. These recommendations are based on the following observations: (1) shorter periods of supine rest significantly reduce the sensitivity of postural vital signs for detecting blood loss, and (2) after normal persons stand, the pulse rate stabilizes after 45 to 60 seconds and the blood pressure stabilizes after 1 to 2 minutes. Counting the heart rate first, beginning at 1 minute, allows more time for the blood pressure to stabilize.

Supine vital signs should always be compared with standing vital signs, because sitting instead of standing significantly reduces the clinician's ability to detect postural changes after blood loss.^{25,26}

F. COMMON ERRORS

Biologic variation of blood pressure is common, and many studies show that blood pressure measurements vary with physical activity, smoking, caffeine ingestion, changes in emotional state, varying temperatures, and different seasons.²⁷⁻²⁹ In addition, the blood pressure measurement may be inaccurate because of inappropriate technique, improper equipment, or other biases related to the observer.^{12,28}

I. WRONG CUFF SIZE

In 1901, von Recklinghausen discovered that Riva-Rocci's original blood pressure cuff, with a bladder about the size of a bicycle tire, was too narrow and often overestimated the true blood pressure, especially in larger arms.^{7,30,31} Subsequent investigations have shown that both the bladder width and length affect the measurement,

although if the bladder encircles at least 80% of the arm's circumference, the effect of width is minimized.^{15,30,32} The bladder of the standard cuff measures 12 × 23 cm and thus is appropriate only for arm circumferences up to 28 cm, which includes only 60% to 70% of the adult population.³⁰

Cuffs that are too short overestimate blood pressure because they transmit cuff pressure inefficiently to the underlying soft tissues. Much higher cuff pressures are then necessary to cause collapse of the artery, leading the clinician to misdiagnose hypertension when it is not present.³² This error is greater the farther the center of the bladder is positioned from the brachial artery.¹⁵

The significance of the opposite error—underestimation of true blood pressure by using a cuff that is too large—is controversial, although most studies show that such an error is small. Table 17.1 presents the mean errors resulting from using cuffs that are too small or too large.³³ These data are based on measurements of blood pressure in the same individual with three cuffs of different sizes, assuming that the most accurate measurement is the one made with the smallest cuff encircling 80% of the arm. The greatest errors, according to these data, occur from using too small of a cuff; the risk of underestimating true pressure with too large a cuff is relatively minor.

2. AUSCULTATORY GAP

Up to 20% of elderly patients with hypertension have an **auscultatory gap**, which means that the phase 1 Korotkoff sounds normally appear at systolic pressure but then disappear for varying lengths of time before they reappear above the diastolic pressure.³⁴ This auscultatory gap is important because inflation of the cuff just to the initial disappearance of sounds (i.e., auscultatory gap) significantly underestimates the true systolic blood pressure. Because the distal pulse persists during the auscultatory gap, however, clinicians can avoid this mistake by palpating the systolic pressure before using the stethoscope.

The cause of the auscultatory gap remains a mystery. Patients with auscultatory gaps have twice as much arterial atherosclerotic plaque as those without a gap,

TABLE 17.1 Blood Pressure Cuff Size and Error in Measurement*

Cuff Bladder Size	ARM CIRCUMFERENCE		
	28 cm or less	29 to 42 cm	43 cm or more
Regular (12 × 23 cm)	Accurate	Overestimates SBP by 4-8 mm Hg DBP by 3-6 mm Hg	Overestimates SBP by 16-17 mm Hg DBP by 10-11 mm Hg
Large (15 × 33 cm)	Underestimates SBP by 2-3 mm Hg DBP by 1-2 mm Hg	Accurate	Overestimates SBP by 5-7 mm Hg DBP by 2-4 mm Hg
Thigh (18 × 36 cm)	Underestimates SBP by 5-7 mm Hg DBP by 1-3 mm Hg	Underestimates SBP by 5-7 mm Hg DBP by 2-4 mm Hg	Accurate

DBP, Diastolic blood pressure reading; SBP, systolic blood pressure reading.

*Overestimation means that hypertension may be diagnosed in someone with normal blood pressure; underestimation means that the blood pressure reading may be normal in someone who actually has high blood pressure. See text for further discussion.

Based on reference 33.

suggesting perhaps that the gap is somehow related to arterial stiffness.³⁴ Venous congestion also seems to promote auscultatory gaps, because slow cuff inflation (which increases venous congestion) may produce an auscultatory gap and elevation of the arm before inflating the cuff may make it disappear.³¹

The auscultatory gap was discovered by Krylov in 1906, one year after Korotkoff's discovery.¹¹ In part, the discovery of the auscultatory gap was responsible for the initial reluctance of clinicians to adopt Korotkoff's method of indirect blood pressure measurement.⁷

3. INAPPROPRIATE LEVEL OF THE ARM

The recommended position of the patient's elbow is the "level of the heart," which is usually regarded as the fourth intercostal space at the sternum. If the patient's arm is instead 6 to 7 cm higher (e.g., level of the sternomanubrial junction), both the systolic and diastolic readings will be about 5 mm Hg lower. If the arm is 7 to 8 cm lower (e.g., level of the xiphosternal junction), the pressures will be about 6 mm Hg higher.³⁵

These errors are completely explained by the hydrostatic effect. When the arm is at the lower position, for example, the measured pressure is the sum of the blood pressure in the artery plus the weight of a column of blood 8 cm high: $8 \text{ cm blood} = (8 \div 13.6) \times 1.06 = 0.6 \text{ cm}$ or 6 mm Hg ($13.6 = \text{density of mercury}$; $1.06 = \text{density of blood}$).

4. TERMINAL DIGIT PREFERENCE (AUSCULTATORY METHOD)^{27,28}

Clinicians tend to round off blood pressure readings to the nearest 0, 5, or other preferred number, a bias called **terminal digit preference**. Clinical studies minimize this and other observer biases by using oscillometric devices or a random zero sphygmomanometer (an instrument that blinds the clinician to the true reading).^{29,36}

G. OTHER VARIABLES

For years, clinicians believed that pressing too firmly with the stethoscope artificially decreased diastolic blood pressure readings, but recent studies show this is not true.³⁷ Whether the bell or diaphragm of the stethoscope are used^{38,39} or whether the stethoscope is placed under the cuff or just outside the cuff³⁷ does not significantly affect the measurement. Raising the patient's arm overhead for 30 seconds before returning it to the normal position and inflating the cuff will intensify Korotkoff sounds without significantly changing the pressure reading.⁴⁰

III. THE FINDINGS AND THEIR CLINICAL SIGNIFICANCE

A. HYPERTENSION

1. ESSENTIAL HYPERTENSION

Essential hypertension is defined as three or more blood pressure readings taken over three visits separated by weeks whose average exceeds 140/90 (i.e., systolic blood pressure of 140 mm Hg and diastolic blood pressure of 90 mm Hg). Detecting essential hypertension is the reason blood pressure should be measured in every person, even when asymptomatic, because the disorder is common and treatable and because treatment reduces cardiovascular morbidity and overall mortality.⁴¹

2. PSEUDOHYPERTENSION AND OSLER SIGN

Pseudohypertension describes the finding of elevated indirect measurements in persons who have normal intra-arterial pressure. The traditional explanation for

pseudohypertension is that the artery under the cuff is so stiff and calcified that it remains open long after the cuff pressure exceeds systolic blood pressure, continuing to produce Korotkoff sounds.

The diagnosis of pseudohypertension requires direct cannulation of the patient's artery, which is of course inappropriate and impractical during daily routine. A single study from 1985 proposed that a simple physical finding, **Osler sign**, accurately identifies patients with pseudohypertension.⁴² This sign is positive if the patient's radial or brachial artery distal to the cuff remains palpable after inflation of the cuff above systolic blood pressure.

Osler sign, however, has limited clinical value. It occurs commonly in elderly individuals, whether or not they have hypertension (presenting in 11% of individuals above the age of 75 years and 44% over the age of 85 years).⁴³ Other investigators have shown that almost all patients with Osler sign do not have pseudohypertension but instead have direct measurements that exceed the indirect ones.^{44,45}

Although pseudohypertension remains an important problem in blood pressure measurements of the legs, especially in diabetic patients with intermittent claudication (see [Chapter 54](#)), undue emphasis on pseudohypertension in the brachial artery misses the point that all clinical studies demonstrating the benefits of treating essential hypertension used the blood pressure cuff and indirect measurements, not intra-arterial ones.

B. HYPOTENSION

In patients with acute illness, hypotension is ominous. It predicts death in patients hospitalized in the intensive care unit (likelihood ratio [LR] = 3.1; [EBM Box 17.1](#)) and in patients with bacteremia (LR = 4.9), pneumonia (LR = 7.6), and myocardial infarction (LR = 15.5). Presumably, it predicts mortality in many other acute disorders as well. The APACHE scoring system, which predicts the risk of hospital mortality among patients in the intensive care unit, assigns more points (and thus a higher risk) to severe hypotension than to any other vital sign or laboratory variable.⁵⁶

Hypotension also predicts adverse outcomes besides death. In patients with myocardial infarction, a systolic blood pressure of less than 80 mm Hg predicts a greater incidence of congestive heart failure, ventricular arrhythmias, and complete heart block.⁵⁴ In patients presenting with syncope, an initial systolic blood pressure of less than 90 mm Hg increases the probability of adverse events in the next 7 days (sensitivity 8% to 18%, specificity 95% to 99%, positive LR 4.2).⁵⁷⁻⁶¹ Finally, in hospitalized patients with a wide variety of problems, low blood pressure readings greatly increase the risk of serious adverse outcomes in the next 24 hours (≤ 90 mm Hg LR = 4.7; ≤ 85 mm Hg LR = 9.0; ≤ 80 mm Hg LR = 16.7; see [EBM Box 17.1](#)).[†]

C. DIFFERENCES IN PRESSURE BETWEEN THE ARMS

The average difference in systolic blood pressure between the two arms is 6 to 10 mm Hg.^{62,63} Differences of 20 mm Hg or more are uncommon and detect obstructed flow in the subclavian artery (i.e., >50% to 60% obstruction) of the arm with the lower pressure (sensitivity 70% to 90%, specificity 99%, positive LR 89.1, and negative LR 0.2).^{64,65} This is a significant finding in two clinical settings: subclavian steal syndrome and aortic dissection.

[†]Two out of three of these adverse outcomes were unexpected transfer to ICU care. Although this suggests circular reasoning (i.e., hypotension was likely a principal reason for transfer), the remaining one out of three adverse events was *unexpected cardiac arrest* or *unexpected death* in the general medicine ward.

**EBM BOX 17.1****Hypotension and Prognosis***

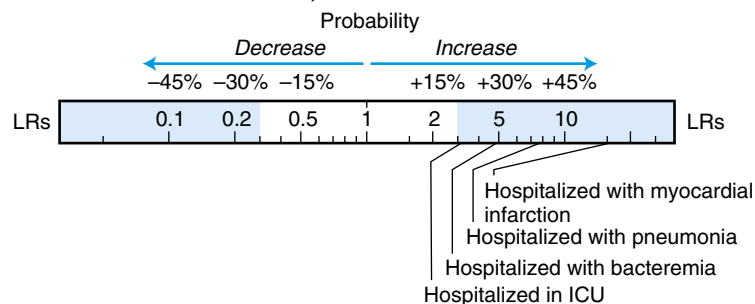
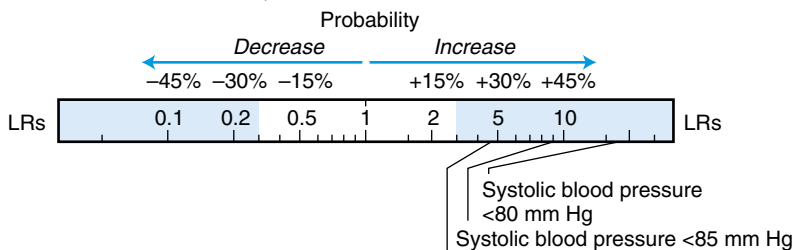
Finding (Reference)	Sensitivity (%)	Specificity (%)	Likelihood Ratio [†] if Finding Is	
			Present	Absent
Predicting Hospital Mortality				
Systolic blood pressure <90 mm Hg				
Patients in intensive care unit ^{46,47}	21-78	67-95	3.1	NS
Patients with bacteremia ^{48,49}	13-71	85-98	4.9	NS
Patients with pneumonia ⁵⁰⁻⁵³	11-41	90-99	7.6	0.8
Systolic blood pressure ≤80 mm Hg				
Patients with acute myocardial infarction ⁵⁴	32	98	15.5	0.7
Predicting Adverse Outcome in Hospitalized Patients ⁵⁵				
Systolic blood pressure ≤90 mm Hg	34	93	4.7	0.7
Systolic blood pressure ≤85 mm Hg	25	97	9.0	0.8
Systolic blood pressure ≤80 mm Hg	21	99	16.7	0.8

*Diagnostic standard: for *adverse outcome*, unexpected cardiac arrest, unplanned ICU admission, or unexpected death.

[†]Likelihood ratio (LR) if finding present = positive LR; LR if finding absent = negative LR.

NS, Not significant.

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HYPOTENSION, PREDICTING MORTALITY**HYPOTENSION, PREDICTING ADVERSE OUTCOME**

I. SUBCLAVIAN STEAL SYNDROME

The finding of one weak radial pulse in a patient with symptoms of vertebral-basilar ischemia (episodic vertigo, visual complaints, hemiparesis, ataxia, or diplopia) suggests **subclavian steal syndrome**. In this syndrome, stenosis or occlusion of one subclavian artery proximal to the origin of the vertebral artery reduces the pressure distal to the obstruction, which causes the flow in the vertebral artery to reverse directions: instead of traveling normally up the vertebral artery to perfuse the brain, blood flow courses downward to perfuse the arm (i.e., the arm *steals* blood from the posterior cerebral circulation).^{66,‡} Ninety-four percent of patients with subclavian steal have a systolic blood pressure that is 20 mm Hg or more, which is lower on the affected arm (the mean difference between the arms is 45 mm Hg in affected patients).⁶⁸ Most patients have an ipsilateral radial pulse that is diminished or absent and a systolic bruit over the ipsilateral subclavian artery.⁶⁸ The left side is affected in 70% and the right side in 30%.⁶⁸

2. AORTIC DISSECTION

The finding of a difference in blood pressure between the two arms in a patient with acute chest pain suggests aortic dissection. **EBM Box 17.2** presents the accuracy of physical examination in over 1400 patients presenting to emergency departments with acute chest or upper back pain suspicious for aortic dissection. In these studies, the presence of a pulse deficit (i.e., absent extremity or carotid pulse; interarm systolic difference >20 mm Hg) increased the probability of aortic dissection (LR = 4.2). Mediastinal or aortic widening on chest radiography also increased the probability of dissection, although only modestly (LR = 2.0); the *absence* of mediastinal widening *decreased* probability (LR = 0.3).^{69,71,73}

In these studies, the murmur of aortic regurgitation was diagnostically unhelpful, possibly because of the highly selected nature of enrolled patients: overall, enrolled patients represented only 0.3% of patients with chest or back pain evaluated in these centers;⁷¹ one-third had the murmur of aortic regurgitation, and half had the diagnosis of dissection eventually confirmed.

von Kodolitsch et al.⁷¹ have identified three independent predictors of aortic dissection in patients with acute chest pain: (1) pain that is tearing or ripping; (2) pulse deficits, blood pressure differentials (>20 mm Hg), or both; and (3) mediastinal or aortic widening on chest radiography. The absence of all three predictors *decreases* the probability of dissection (LR = 0.1; see **EBM Box 17.2**); two predictors increase the probability of dissection (LR = 5.3); and the presence of all three predictors is pathognomonic for dissection (LR = 65.8).

Rare patients with aortic dissection present with the physical findings of pulsatile sternoclavicular joints⁷⁴ or unilateral femoral pistol shot sounds (see **Chapter 45**).⁷⁵

In patients with established aortic dissection, three findings increase the probability that the dissection involves the proximal aorta (i.e., it is a type A dissection, not a type B dissection): systolic blood pressure less than 100 mm Hg (LR = 5), murmur of aortic regurgitation (LR = 5), and a pulse deficit (LR = 2.3).^{69,70,74,76,77} In patients with acute type A dissection, pulse deficits are associated with increased hospital mortality.⁷⁸

‡ An excellent online video of vertebral retrograde flow in a patient with subclavian stenosis is available in the supplementary material provided by Aithal and Ulrich.⁶⁷



EBM BOX 17.2
*Aortic Dissection**

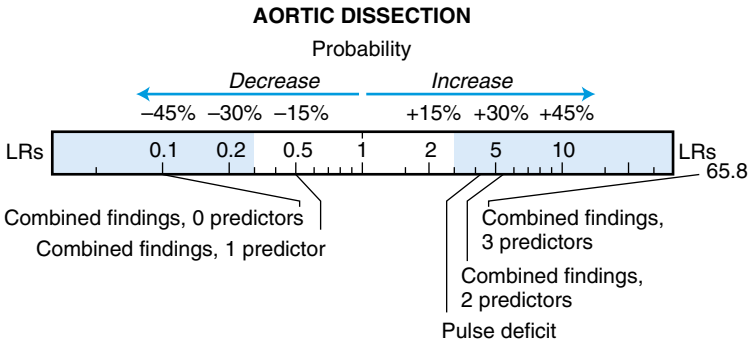
Finding (Reference) [†]	Sensitivity (%)	Specificity (%)	Likelihood Ratio [‡] if Finding Is	
			Present	Absent
Individual Findings				
Pulse deficit ^{69,72}	12-49	82-99	4.2	0.8
Aortic regurgitation murmur ^{69,73}	5-49	45-95	1.5	NS
Focal neurologic signs ^{71,72}	14-20	93-100	NS	0.9
Combined Findings⁷¹				
0 predictors	4	47	0.1	—
1 predictor	20	—	0.5	—
2 predictors	49	—	5.3	—
3 predictors	27	100	65.8	—

*Diagnostic standard: for *aortic dissection*, transesophageal echocardiography,^{69,73} aortography,⁷⁰ or any of a variety of tests (i.e., computed tomography, magnetic resonance imaging, transesophageal echocardiography, or digital angiography).^{71,72}

[†]Definition of findings: for *pulse deficit*, absent extremity or carotid pulse^{69,70} or 20 mm Hg difference in blood pressure in the arms, absent extremity or carotid pulse, or both;^{71,72} for *combined findings*, see text.

[‡]Likelihood ratio (LR) if finding present = positive LR; LR if finding absent = negative LR. NS, Not significant.

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D. DIFFERENCES IN PRESSURE BETWEEN ARMS AND LEGS

This finding is valuable in two clinical settings:

I. CHRONIC ISCHEMIA OF THE LOWER EXTREMITIES

[Chapter 54](#) describes calculation of the ankle-arm index, which is the principal bedside tool used in patients with intermittent claudication.

2. COARCTATION OF THE AORTA

In young patients with hypertension, the finding of an unobtainable blood pressure in the legs or a blood pressure that is much lower in the legs than arms suggests the diagnosis of coarctation of the aorta.^{79,80} These patients also have hypertension of the arms (96% have a blood pressure >140/90), femoral pulses that are absent or diminished and delayed (100%), augmented carotid pulsations, various murmurs (usually a systolic murmur at the sternal border and a continuous murmur posteriorly over the upper spine), and visible collateral arteries (usually around the scapula, intercostal spaces, or axilla).^{79,80}

During simultaneous palpation of the femoral and radial arteries of healthy persons, it is impossible to tell which comes first. In patients with coarctation, however, the femoral pulse is delayed, due both to delay in arrival at the legs and to more rapid than normal conduction of the wave to the arms.⁸¹

In one study of 1206 children with unexplained heart murmurs, clinicians correctly diagnosed coarctation of the aorta in 18 of 22 affected patients. (In this study, the overall accuracy for detecting coarctation by bedside examination—presumably using arm-to-leg blood pressure or pulse discrepancies—was sensitivity of 82%, specificity of 100%, positive LR = 242, and negative LR = 0.2.)⁸²

E. ABNORMAL PULSE CONTOUR

The three abnormalities of pulse contour—pulsus paradoxus, pulsus alternans, and pulsus bisferiens—are easily detectable with the blood pressure cuff using the auscultatory method (see [Chapter 15](#)).

F. ABNORMAL PULSE PRESSURE

I. ABNORMALLY SMALL PULSE PRESSURE

Since the pulse pressure depends on stroke volume, clinicians have tried for decades to use it as a way to quantify cardiac output. This relationship has been validated in one setting: patients with known left ventricular dysfunction. In these patients, the finding of a proportional pulse pressure less than 0.25 (proportional pulse pressure = pulse pressure divided by systolic pressure) detects a cardiac index less than 2.2 L/min/m² with a sensitivity of 70% to 91%, specificity of 83% to 93%, positive LR = 6.9, and negative LR = 0.2.^{83,84}

In contrast to conventional teachings, many patients with significant aortic stenosis have a normal pulse pressure (see [Chapter 44](#)).⁸⁵ [Chapter 70](#) discusses using changes in pulse pressure after passive leg elevation as a sign of volume responsiveness in critically ill patients.

2. ABNORMALLY LARGE PULSE PRESSURE

In patients with the murmur of aortic insufficiency, a pulse pressure of 80 mm Hg or more increases the probability that the regurgitation is moderate or severe, with a sensitivity of 57%, specificity of 95%, and positive LR = 10.9.⁸⁶

G. ORTHOSTATIC HYPOTENSION

When a person stands, 350 to 600 mL of blood shifts to the lower body. Normally, the blood pressure remains relatively stable during this shift because of compensatory increases in cardiac output, heart rate, and systemic vascular resistance, and transfer of blood from the pulmonary circulation to the systemic side.²⁴ Orthostatic hypotension, usually defined as a fall in systolic blood pressure of 20 mm Hg or more when the patient stands from the supine position, may occur if (1) compensatory mechanisms fail (i.e., autonomic insufficiency), or (2) the patient has lost excessive amounts of fluid from the vascular space (e.g., acute blood loss).

TABLE 17.2 Vital Signs and Acute Blood Loss*

Physical Finding (Reference)	Moderate Blood Loss, Sensitivity (%)	Large Blood Loss, Sensitivity (%)	Specificity (%)
Postural pulse increment ≥ 30 /min or severe postural dizziness ^{25,87-89}	7-57	98	99
Postural hypotension (≥ 20 mm Hg decrease in SBP) ^{87,88}	9	—	90-98
Supine tachycardia (pulse > 100 /min) ^{26,89-92}	1	10	99
Supine hypotension (SBP < 95 mm Hg) ^{26,90,91,93,94}	13	31	98

*Data obtained from 568 normal persons, mostly young and healthy, after “moderate” blood loss (phlebotomy of 450-630 mL) or “large” blood loss (phlebotomy of 630-1150 mL). “Specificity” from same patients when euvoletic, before blood loss. Results are overall mean frequency or, if statistically heterogeneous, the range of values.

†Definition of finding: For *postural*, the difference between supine and standing measurements; for *postural hypotension* (≥ 20 mm Hg decrease in SBP), the finding applies only to patients able to stand without severe dizziness.

SBP, Systolic blood pressure.

Based upon reference 24.

1. POSTURAL VITAL SIGNS IN HEALTHY PERSONS

As normovolemic persons stand up from the supine position, the pulse increases on average by 10.9 beats/minute, systolic blood pressure decreases by 3.5 mm Hg, and diastolic blood pressure increases by 5.2 mm Hg.²⁴ Postural hypotension, defined as a decrement in systolic blood pressure of 20 mm Hg or more, occurs in 10% of normovolemic individuals younger than 65 years and in 11% to 30% of normovolemic individuals older than 65 years.²⁴ As persons age, the postural pulse increment diminishes ($r = -0.50$, $p < 0.02$); this phenomenon and the observation that older persons have more postural hypotension suggest that autonomic reflexes decline as persons age.

2. VITAL SIGNS AND HYPOVOLEMIA

Table 17.2 presents the vital signs from normal persons before and after phlebotomy of 450 to 630 mL (moderate blood loss) or 630 to 1150 mL (large blood loss).[§] Chapter 11 reviews the other physical findings of hypovolemia.

A. POSTURAL CHANGE IN PULSE

Table 17.2 shows that the most valuable observation is *either* a postural pulse increment of 30/minute or more *or* the inability of the patient to stand long enough for vital signs because of severe dizziness. Virtually all persons have one or both of

[§]Calculating LR for these data is not appropriate, because *acute blood loss* has endless gradations of severity, many of which are important to the clinician. For example, the LR of physical signs for moderate blood loss are of little use to the clinician who, when taking care of the patient with melena, regards blood loss of 400 mL (*disease-negative* according to the LR) to be as significant as a loss of 500 mL (*disease-positive*). Table 17.2 instead just illustrates the general trends of vital signs with increasing amounts of blood loss.

these findings after large amounts of blood loss (sensitivity = 98%), but only 1 in 5 persons develop either of them after moderate blood loss (sensitivity ranges from 7% to 57%; Table 17.2).²⁴ These findings are durable after hemorrhage, lasting at least 12 to 72 hours if intravenous fluids are withheld.^{26,95,96}

B. POSTURAL CHANGE IN BLOOD PRESSURE

After excluding those patients unable to stand for vital signs (which includes almost all patients after large amounts of blood loss), the finding of postural hypotension (a postural decrement in systolic blood pressure of 20 mm Hg or more) has no proven value, being found just as often in patients before blood loss as after it. For example, in persons younger than 65 years, postural hypotension is found in 8% before moderate blood loss and 9% after blood loss. For those 65 years or older, postural hypotension is detected in 11% to 30% before blood loss and about 25% after blood loss.^{24,28}

Obviously, because severe dizziness with standing is a valuable finding but the measured postural hypotension of 20 mm Hg is inaccurate, there must be an intermediate level of postural fall (e.g., 30 mm Hg, 40 mm Hg, or another value), not yet identified, that better discriminates between patients with and without blood loss.

C. SUPINE PULSE AND SUPINE BLOOD PRESSURE

In patients with suspected blood loss, both supine tachycardia and supine hypotension are specific indicators of significant blood loss, although both findings are infrequent. After moderate blood loss, 1% have tachycardia in the supine position and only 13% have supine hypotension; after large blood loss, only 10% have tachycardia and 31% have hypotension.

Sinus bradycardia, in contrast, is a common arrhythmia after blood loss and frequently precedes the drop in blood pressure that causes patients to faint.²⁴

H. BLOOD PRESSURE AND IMPAIRED CONSCIOUSNESS

Patients with impaired consciousness may have either a structural intracranial lesion (e.g., stroke or brain tumors) or metabolic encephalopathy (e.g., hepatic encephalopathy, diabetic coma, drug intoxication, or sepsis). Patients with structural lesions tend to have higher blood pressures (from reflex responses to increases in intracranial pressure—the Cushing reflex—or from the etiologic association of hypertension and stroke) than do patients with metabolic encephalopathy (whose severe comorbidities often are associated with lower blood pressure). In two studies of consecutive patients with impaired consciousness (i.e., Glasgow coma scale less than 15) but no history of head trauma, a systolic blood pressure of 160 mm Hg or more significantly increased the probability of a structural lesion (LR = 7.3; *EBM Box 17.3*).

I. CAPILLARY FRAGILITY TEST (RUMPEL-LEEDE TEST)

Traditionally, the blood pressure cuff was used to test capillary fragility, although measurements of blood pressure were not part of the test. Capillary fragility tests were designed to detect abnormally weakened capillary walls in the skin that would burst more easily when distended, resulting in the appearance of high numbers of petechiae. A large number of diseases were associated with capillary fragility, ranging from coagulopathies, vitamin deficiencies (e.g., scurvy), infectious diseases (e.g., scarlet fever), and endocrine disorders (e.g., hyperthyroidism), to dermatologic disorders (e.g., Osler-Weber-Rendu syndrome).⁹⁹

Both negative and positive pressure methods were used. The negative pressure technique applied suction to a defined area of the skin, a technique whose undoing



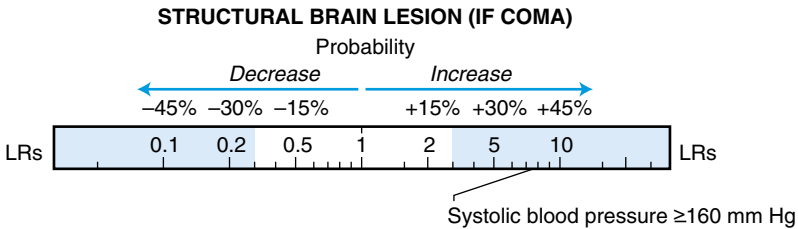
EBM BOX 17.3

Systolic Blood Pressure and Impaired Consciousness

Finding (Reference)	Sensitivity (%)	Specificity (%)	Likelihood Ratio* if Finding Is	
			Present	Absent
Detecting Structural Brain Lesion				
Systolic blood pressure ≥160 mm Hg ^{97,98}	37-58	93-94	7.3	0.6

*Likelihood ratio (LR) if finding present = positive LR; LR if finding absent = negative LR.

[Click here to access calculator](#)



was the eventual demonstration that the number of resulting petechiae depended on not only the age of the patient but also on the time of day, season, and psychic influences.¹⁰⁰ Positive pressure methods, introduced at the turn of the century by Drs. Rumpel and Leede, consisted of raising the venous pressure by a tourniquet or blood pressure cuff around the arm and counting petechiae that subsequently developed in a defined area distally. This test was eventually standardized,¹⁰⁰ but interest fell after the introduction of better diagnostic tests for coagulation and the other associated disorders. More recently, increased capillary fragility was believed to represent a sign of diabetic retinopathy,¹⁰¹ but this was soon disproven.¹⁰²

Nonetheless, a variation of this Rumpel-Leede test (called the **tourniquet test****) remains important in the developing world as a diagnostic test for dengue fever and its complications. In patients with undifferentiated fever presenting to clinicians working in tropical settings, a positive tourniquet test detected confirmed dengue infection with a sensitivity of 34% to 68%, specificity of 84% to 99%, and positive LR of 6.8.¹⁰³⁻¹⁰⁶

The references for this chapter can be found on www.expertconsult.com.

**In the standard method of the tourniquet test, the clinician inflates the blood pressure cuff midway between systolic and diastolic blood pressure for 5 minutes and then counts the number of petechiae that form in a 2.5-cm² area just distal to the antecubital fossa. The positive test is 20 petechiae or more.

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